CASE REPORTS

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Tuberculous Otomastoiditis: An Old Disease Renewed

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INFECTION OF THE MIDDLE EAR is a familiar diagnosis to pediatricians. As many as 84 percent of children have otitis media at some point during infancy or childhood.[1] The usual acute pathogens are also well known to physicians. It is not so well appreciated, however, that *Mycobacterium tuberculosis* is still occasionally associated with middle ear suppuration.

Tuberculous otitis media was first described in the early 19th century, long before Koch's discovery of tubercle bacilli in 1882.[2] In the first three decades of this century, otic tuberculosis accounted for 2 percent to 15 percent of pediatric suppurative otitis.[3,4] With sterilization of milk and advances in the diagnosis, prevention and therapy of tuberculosis, the incidence of tuberculous otomastoiditis has become rare in the United States. Tuberculosis of the middle ear does, however, continue to occur, particularly among immigrants from less developed countries, and numerous cases have been reported in this country in the past 20 years.[5-13] The following cases illustrate the characteristic features of the disease.

Reports of Cases

CASE 1. A 16-year-old Mexican-American boy was referred to the University of California, Los Angeles, outpatient department by the school nurse for evaluation of a hearing loss. The patient had immigrated to the United States from Mexico in the past year. He had reportedly been healthy until 5 years of age, when a large pimple was noted over the area of the right mastoid. The lesion drained purulent material and resolved in several weeks. Meanwhile, a thick, green, odorless fluid began to drain from the right ear canal, which continued to drain intermittently for the next 11 years, in spite of multiple courses of both local and oral antibiotic therapy. Hearing loss was first noted by teachers when the boy was 7 years old.

A physical examination showed a well-developed adolescent with a purulent, greenish, odorless drainage observed in the right external auditory canal. The periphery of the drum was scarred and moderately vascularized, and a large perforation of the right tympanic membrane was noted. The malleus was not visualized, and exuberant "popcorn-like" tissue was noted in the superior-anterior portion of the field. A standard 5 TU (tuberculin units) purified protein derivative (PPD) tuberculin skin test was positive with 20 mm induration at 48 hours.

A 20 to 55 dB hearing loss in the right ear was noted, and audiometry confirmed unilateral incapability of auditory discrimination at all frequencies with total right-sided deafness at frequencies greater than 1,000 Hz. A roentgenogram of the chest showed a calcified granuloma in the upper right lung field and mastoid films disclosed sclerosis of the right mastoid. Temporal bone tomograms indicated an absence of distinct middle ear structures, but showed general sparing of the inner ear. Culture of the ear drainage on Löwenstein-Jensen medium grew *M tuberculosis,* sensitive to all tested antituberculosis agents.

Treatment with INH (isonicotinic acid hydrazide; also called isoniazid), rifampin, and ethambutol was initiated, and on follow-up examination six months later the patient was asymptomatic. Repeat audiometric testing showed a profound hearing deficit in the right ear. Both parents and four siblings also had positive intermediate (5 TU) tuberculin skin tests, and they all were treated with INH for a year. Although no active tuberculosis was found in the family, both parents had roentgenograms that suggested previous intrathoracic tuberculosis.

CASE 2. A 6-year-old Mexican-American girl

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was seen in the outpatient clinic with a seven-month history of limping of the right leg. On admission to hospital for workup of possible tuberculous osteomyelitis, it was noted that she had a green, odorless fluid draining from the left ear, in addition to a perforation of the left tympanic membrane. *Mycobacterium tuberculosis* was isolated from both the otic drainage and the bone. Her intermediate strength (5 TU) tuberculin skin test measured 20 mm of induration and an x-ray study of the chest showed an apical left lower lobe infiltrate consistent with tuberculosis. The child had severe hearing loss on audiologic screening. INH and rifampin therapy was initiated. When she reappeared six months later the otorrhea had stopped, and culture of the ear for *M tuberculosis* was negative. Four of her nine siblings and both parents had positive (5 TU) tuberculin skin tests.

Discussion

Tuberculosis remains a cause of significant morbidity and mortality.[7] Although effective antituberculous therapy has long been available, recognition of extrapulmonary tuberculosis continues to be difficult.[2] Indeed, the failure to consider this disease in the absence of respiratory symptoms may explain the unchanging incidence of extrapulmonary tuberculosis in recent decades, despite the decline in active pulmonary tuberculosis during that period.[8] It is important that physicians in the western United States be aware that persons of Spanish-American and American Indian descent have a significantly higher incidence of extrapulmonary tuberculosis.[8,9]

Because tuberculous otomastoiditis is relatively rare, the diagnosis is seldom considered in cases of chronic otitis media, and many cases progress unrecognized for prolonged periods, as exemplified in the two cases above. Both cases illustrate the usual features of this disease, which include the following: (1) painless otorrhea, (2) a rapidly enlarging perforation of the tympanic membrane (the initial stage of multiple perforations has generally progressed to coalescence by the time of presentation), (3) early and profound hearing deficiency, (4) abundant granulation tissue with necrosis of the ossicles and temporal bone, (5) an infectious process unresponsive to therapy for acute otitis media and (6) a positive tuberculin skin test.

Fever is uncommon in uncomplicated cases. Regional adenopathy is occasionally present and may be pronounced in infants.[10,14] Tuberculous otitis media in children is often associated with complications and tuberculous involvement of the mastoid is not uncommon.[2,3] Hearing loss occurs early in the course of this disease. Involvement of the labyrinth is also common and may be partially responsible for the profound hearing loss, but vestibular dysfunction is rare, presumably because the destruction is a gradual process.[9,11] A characteristic complication in infants is facial nerve palsy, a process which often resolves as the otitis is treated.[7,9] The most serious complication is tuberculous extension to the meninges, which may occur at any time in the course of the otomastoiditis.

Diagnosis of tuberculous otomastoiditis requires a high index of suspicion. Painless otorrhea in a patient with tuberculous infection elsewhere should be considered tuberculous otitis until proved otherwise. The clinical features as described above that occur in any patient should alert physicians to a possible tuberculous cause of the condition, particularly if the patient has lived in a low socioeconomic environment or has immigrated from a foreign country where tuberculosis is endemic.

Mastoid films may disclose destruction of temporal bone and well-pneumatized air-cells, but are not specific for tuberculosis.[2,4] A positive (10 mm or greater induration) 5 TU skin test may be helpful, but a negative test does not rule out tuberculosis.[11] Acid-fast bacilli observed on Ziel-Neelsen stain of otorrheal fluid is strong presumptive evidence of a tuberculous cause.[10,15] Confirmation of the diagnosis of tuberculosis is made by culture of the otic discharge, and microbial sensitivity testing is necessary for proper pharmacotherapy.[3,6] Multiple specimens are advisable because the number of organisms present may vary.

An essential component of management is to search for the primary focus of tuberculous infection. Primary infection may occur in infants and young children drinking unpasteurized cow's milk or nursing from women with generalized tuberculosis, with subsequent transport of *M tuberculosis* organisms by way of the eustachian tube. This presentation is rare in the United States today. Secondary infection, the usual mechanism of tuberculous otomastoiditis, results from hematogenous spread to the mastoid or via the eustachian tube through transport of autocontaminated sputum. The primary focus may resolve before presentation, as was the case in our first patient.

Household members should have tuberculin skin tests done as well because the initial contact is usually an adult in the household.

In view of the permanently destructive nature of the disease process, it is important to initiate therapy as soon as possible, with three first-line antituberculosis medications (such as INH, ethambutol, rifampin and streptomycin). Chemotherapy may be altered when sensitivities are determined; however, two-drug therapy is warranted for at least a year. Adherence to a therapeutic regimen of appropriately selected antibiotics effectively halts the disease process. Following successful pharmacotherapy, surgical intervention may be considered for many patients, particularly those who had a prolonged course of illness before effective treatment.[16] Necrotic tissue may be removed via a mastoidectomy.

Tuberculous otitis is a condition readily amenable to appropriate antituberculous chemotherapy. In any otitis which does not respond to the usual antibiotic therapy, or which presents as a painless otorrhea, tuberculosis must be suspect, and PPD testing should be routinely done in these patients. Most of the hearing defict present at the time of initial treatment will be permanent, but further loss can be prevented and progression to a life-threatening meningitis aborted.

Summary

Tuberculosis is a rare cause of middle ear suppuration in the United States today, although with an increase in foreign travel and migration there has been a recent rise in the number of cases, especially of extrapulmonary tuberculosis. In patients presenting with chronic, painless otorrhea or chronic otitis media that does not respond to the usual antibiotic therapy, tuberculous otitis should be considered. The diagnosis is important as tuberculous otomastoiditis is a rapidly progressive, irreversibly destructive process, and effective therapy should be instituted early. The two cases described above represent the characteristic features of this disease and illustrate the severe hearing deficiencies that can result in children. Tuberculous otitis media has been associated with serious consequences, including rapidly progressive permanent deafness and progression to fatal meningitis.

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Hypercalcemia, Pneumothorax and Pneumoperitoneum in a Patient With Pulmonary Mycobacteriosis and Esophageal Carcinoma

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ALTHOUGH CARCINOMA of the esophagus remains one of the least common of all malignant conditions to afflict people in the United States, its prevalence and age-adjusted death rate has been rising slowly but steadily over the past 50 years, particularly among nonwhite Americans. Based on death rates from the National Cancer Institute's Surveillance, Epidemiology and End Results (SEER) program (1973 through 1976), the esti-

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